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REVIEW

Constipation in critically ill adults

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Abstract

Constipation is a common yet underrecognized gastrointestinal complication among critically ill adults, significantly impacting morbidity, length of stay, and overall prognosis. This narrative review explores the current understanding of constipation in the critical care setting, emphasizing the challenges in its definition and identification due to variability in clinical presentation and lack of standardized diagnostic criteria. We examine contributing factors such as immobility, opioid use, altered fluid and electrolyte balance, and the effects of critical illness itself on gastrointestinal motility. Furthermore, we discuss available and emerging management strategies in critically ill adults, including pharmacologic and non-pharmacologic interventions, and highlight the importance of early identification and targeted therapy in improving patient outcomes. Finally, we address the prognostic implications of constipation in critically ill adults and the need for prospective studies to better define its impact and inform evidence-based guidelines. This review aims to raise awareness and stimulate further research into this often-overlooked aspect of gastrointestinal pathophysiology in the intensive care unit.

Key Words: Constipation; Gastrointestinal motility; Bowel dysfunction; Critical illness; Intensive care unit

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Core Tip: Constipation is a frequently overlooked complication in critically ill adults that adversely affects clinical outcomes. This review synthesizes current evidence on its complex pathophysiology, multifactorial etiology and diagnostic challenges within the intensive care unit. It underscores the importance of timely recognition and comprehensive management strategies combining both therapeutic and preventive strategies. By highlighting gaps in standardized diagnostic criteria and therapeutic protocols, this article advocates for further research to establish evidence-based guidelines aimed at improving patient prognosis and quality of care in the intensive care unit.

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INTRODUCTION

Constipation is a common yet underrecognized gastrointestinal complication in critically ill adults[1]. It has been associated with increased rates of infection, morbidity, mortality, prolonged mechanical ventilation and prolonged intensive care unit (ICU) stay, with implications for healthcare costs[2]. Patients attending post-intensive care follow-up clinics have reported constipation as a particularly distressing aspect of their ICU experience[3]. The approach to constipation in the ICU, however, remains inconsistent due to the lack of a universally accepted definition and standardized treatment pathways. There is a lack of consensus in prophylactic laxative use, with inconsistent im-plementation of standardized bowel protocols. Furthermore, management priorities are often directed towards stabilizing vital functions and managing acute illnesses, with supportive measures such as bowel care receiving relatively less attention[4]. To highlight the importance of constipation in critically ill adults for clinicians, this narrative review therefore aims to provide a succinct overview that addresses challenges in its identification, contributing factors, associated clinical consequences, prognostic implications, current management strategies, and future directions.

EPIDEMIOLOGY AND DEFINITION OF CONSTIPATION IN THE CRITICAL CARE SETTING

The incidence of constipation in the ICU varies widely, ranging from 20% to as high as 83% among critically ill adults[5]. Such variation can be attributed to the lack of a standardized definition of constipation in the intensive care setting, despite recommendations from international guidelines and ICU-based studies.

Among general medical patients, Rome IV diagnostic criteria define functional constipation as the presence of two or more of the following six symptoms: (1) Straining to evacuate in more than 25% of the defecations; (2) Lumpy or hard stools in more than 25% of defecations; (3) Fewer than three spontaneous bowel movements per week; (4) Sensation of incomplete evacuation in more than 25% of defecations; (5) Sensation of anorectal blockage in more than 25% of defecations; and (6) Need for manual maneuvers to facilitate more than 25% of defecations. Additionally, symptom-onset must precede diagnosis by at least 6 months, with symptoms persisting for at least three months[6].

However, the Rome IV criteria have limited applicability in the ICU setting. In addition to requiring a prolonged duration of symptoms for diagnosis, these criteria are intended for identifying functional constipation in the absence of secondary causes such as mechanical obstruction, medication effects, or systemic illness - factors that are commonly present in critically ill adults[6]. Furthermore, these criteria rely heavily on patient-reported symptoms, which are difficult to assess in critically ill adults who are delirious, sedated, or mechanically ventilated[6]. Even when patients can communicate, discrepancies may arise when patient-reported symptoms are compared to clinicians' assessments due to the subjective nature of symptoms, differing opinions on their relative importance, and heterogeneous presentations[7]. For instance, while patients may emphasize straining or hard stools, physicians require at least two of six symptoms outlined in the Rome IV criteria to establish a diagnosis of constipation[6]. Moreover, constipated patients may present with upper gastrointestinal symptoms such as bloating, abdominal distension, postprandial fullness, nausea, vomiting and regurgitation, which are not included within the Rome IV criteria[8]. Consequently, individuals reporting symptoms of constipation may not meet formal diagnostic thresholds, while differing perceptions between patients and clinicians may delay the identification, documentation and management of constipation[7,9].

Considering the limitations of Rome IV criteria in critically ill adults, several ICU-based studies have adopted alternative definitions of constipation, primarily based on objective measures such as bowel movement frequency, need for therapeutic interventions, or a combination of clinical signs, as illustrated in Table 1[10-17]. A Working Group on Abdominal Problems from the European Society of Intensive Care Medicine (ESICM) proposed replacing the term "constipation" with "paralysis of the lower gastrointestinal tract", where clinical signs include the absence of defecation for three or more consecutive days without mechanical obstruction[18]. The ESICM supports the three-day cut-off as a defining threshold, as it is commonly used in most epidemiological studies involving ICU patients.

Despite the ESICM recommendations, there remains lack of consensus among ICUs, and many criteria continue to vary considerably in cut-offs, specificity and underlying rationale. ICU-specific definitions have greater pragmatic applicability than the Rome IV criteria, but they have not been formally validated, limiting their diagnostic accuracy. Consequently, inconsistencies remain in the reported incidence and clinical management of constipation. It is generally

Table 1 Key definitions of constipation reported in the intensive care unit

Ref.	Definition of constipation used in the study	Setting	Remarks
Drossman *et al* [10], 2016	≥ 2 of 6 symptoms for ≥ 3 months, symptom onset ≥ 6 months prior (Rome IV criteria)	General population	Not validated in ICU patients; relies on patient self-report and chronic symptom history, which limits applicability in critical illness
Gacouin *et al* [11], 2010	Absence of bowel movement for ≥ 6 days after ICU admission	Medical ICU	Utilizes a longer bowel movement interval threshold compared to most ICU studies
Guardiola *et al* [12], 2016	Paralysis of the lower gastrointestinal tract	ICU	Proposes an alternative term to constipation in an ICU setting
Lewis and Heaton[13], 1997	Types 1-2 (hard, lumpy) suggest constipation (Bristol Stool Form Scale)	General population	Widely used to assess stool consistency; may serve as a complementary tool for assessing constipation in ICU patients
Mostafa *et al* [14], 2003	Failure for the bowel to open for 3 consecutive days	Surgical/medical ICU	Definition applied within a broader investigation of gastrointestinal dysfunction in ICU patients
Nassar *et al*[15], 2009	Need for treatment with laxatives or enemas	ICU	Defined by the need for pharmacological intervention
Patanwala *et al* [16], 2006	Absence of bowel movement within the first 4 days of ICU admission	Medical ICU	Adopts a 96-hour interval instead of the 72-hour period typical for ambulatory patients, accounting for the first 24 hours dedicated to patient stabilization, which can delay initiation of enteral nutrition or bowel regimen
van der Spoel *et al*[17], 2006	First defecation occurring after 6 days	Mixed ICU	One of the earliest studies proposing an ICU-specific operational definition of constipation

ICU: Intensive care unit.

agreed, however, that bowel sounds are unreliable as diagnostic indicators and should not be included in diagnostic criteria[18]. Development of a more consistent and clinically relevant definition of constipation, informed by the specific challenges and priorities of critical care, may enhance diagnostic accuracy and guide timely intervention.

CONTRIBUTING FACTORS TO CONSTIPATION IN CRITICALLY ILL ADULTS

The etiology of constipation in critically ill adults is often multifactorial[19]. Constipation may result from the interplay of critical illness, use of concurrent constipating medications (*e.g.*, high-dose opioids), low-fiber diets, immobility and other contributing factors (Table 2)[20-22]. Understanding the factors particularly prevalent among this population is essential for the early identification of at-risk individuals, allowing for timely and effective intervention. For instance, systematic identification of at-risk individuals using methods such as the Norgine Risk Assessment Tool[23] helps trigger early preventative strategies, which may then accelerate the recovery of critically ill adults[24].

Patient-related factors

Advanced age is a well-established risk factor that impairs gastrointestinal motility, due to age-related reductions in colonic motility, anorectal sensation, and enteric nervous system responsiveness[21]. This is particularly relevant in the ICU, where adults aged ≥ 65 years constitute a substantial and growing proportion of the critically ill adult population[25, 26]. Abdominal surgery is another key contributor to postoperative ileus, characterized by a transient reduction or absence of peristalsis in the immediate postoperative period. Proinflammatory stimuli - including bowel trauma, hypoxia, hypoperfusion, ischemia-reperfusion, and infection - can induce gut injury and motility disturbances[27]. Hypoxia leads to gut mucosal acidosis and gut barrier injury, leading to leakage of gut luminal content, local inflammation, and subsequent systemic inflammatory response[28]. Surgical stress triggers multiple inflammatory pathways, resulting in the release of cytokines, prostanoids, nitric oxide, and reactive oxygen species[29]. Postoperative ileus may be further exacerbated by factors such as reduced oral intake, prolonged immobility, and psychological disorders including anxiety or depression[30].

Immobility, resulting from critical illness, injury, or sedation, is also a significant contributor to constipation among ICU patients. Most critically ill adults are confined to bed and are maintained in supine position, which limits the natural impact of gravity on evacuation. Furthermore, effective defecation requires an increase in intra-abdominal pressure; however, this process is often compromised in patients lying on bed due to restricted mobility. Prolonged bed rest also may weaken the abdominal wall muscles, impairing the ability to generate sufficient intra-abdominal pressure necessary for bowel movements[23].

Disease-related factors

Critically ill adults frequently present with multiple comorbid conditions and varying degrees of organ dysfunction or

failure. Certain systemic illnesses can predispose patients to constipation either through direct impact on gastrointestinal physiology or through broader impact on overall health, such as reduced mobility or frailty[23]. Acute kidney injury, for example, has been associated with an increased risk of constipation, potentially due to the accumulation of inflammatory mediators and metabolic waste products within the gastrointestinal tract[24,31]. Sepsis and shock, both prevalent in ICU settings, can impair gastrointestinal motility through elevated levels of circulating endotoxins, pro-inflammatory cytokines, inducible nitric oxide production and splanchnic hypoperfusion[15,17]. Metabolic disorders such as diabetes mellitus type 2 are also associated with constipation. While the precise mechanisms are not fully elucidated, it has been proposed that peripheral neuropathy, including that affecting lower limbs, may be correlated with autonomic neuropathy of the intestinal tract[32,33]. Electrolyte imbalances common in critically ill adults, including hypokalemia, hypercalcemia, and hypomagnesemia, can directly affect gut motor function by altering nerve conduction and impair smooth muscle contraction, predisposing to constipation[27,34]. Such pathophysiological changes arising from systemic illnesses can disrupt the nutritional, protective, immune, and endocrine functions of the gastrointestinal tract, contributing to gastrointestinal dysmotility[24].

Neurological diseases are also key contributors to constipation. New-onset constipation is a common complication following acute stroke in critically ill adults, particularly among those with a history of pre-existing constipation[35]. Stroke-related injury may disrupt the central autonomic regulation of gut motility, leading to sympathetic overactivity and reduced colonic propulsion. This is thought to occur *via* reduced expression of neuronal nitric oxide synthase in myenteric neurons, which is essential for smooth muscle relaxation and coordinated colonic transit[36]. In addition, many stroke patients exhibit impaired anorectal sensation, such as elevated rectal sensory thresholds, which is associated with post-stroke constipation[37]. New-onset constipation has been associated with poorer outcomes in patients experiencing strokes of moderate severity[35]. Beyond stroke, other neurological insults and critical illness can also impair gastrointestinal function *via* the brain-gut-microbiota axis, a complex network linking the central and enteric nervous systems with the intestinal microbiota[38]. The brain may regulate the intestinal microbiota through two main pathways: (1) *Via* the autonomic nervous system, which modulates intestinal motility, transit, secretion and permeability; and (2) *Via* luminal hormone secretion, which can directly influence microbial gene expression[39]. Disruption of this axis - whether due to neurological injury, inflammation, stress, or critical illness - can impair motility, visceral sensitivity, and secretion patterns, all of which may contribute to constipation in ICU patients[38]. Critical illness itself may further induce intestinal dysbiosis which is implicated in gut motility disorders and impaired barrier function[40]. Reduced abundance of short-chain fatty acid-producing bacteria may impair colonocyte energy metabolism and motility[41].

Medication-related factors

Polypharmacy is a common feature in critically ill adults, often arising from the need to manage multiple organ dysfunctions and failure[42]. These patients frequently receive concomitant medications, including opioids, sedatives, antihypertensives and diuretics - many of which cause colonic hypomotility[43]. The extensive use of such pharmacological agents has been associated with an increased risk of constipation and greater reliance on laxatives[44].

Opioid-induced constipation (OIC), in particular, is a well-recognized complication occurring in up to 83% of critically ill adults, due to the effects of opioids on both the central nervous system and gastrointestinal tract[14,45]. The high density of peripheral - opioid receptors in the enteric system mediates the majority of opioid-induced gastrointestinal effects, including inhibition of intestinal peristalsis, delayed gastric emptying, increased fluid absorption, and reduced intestinal transit time[46,47]. This is supported by the efficacy of peripherally acting-opioid receptor antagonists, such as methylnaltrexone, which reverse OIC without influencing central analgesia[48,49]. Opioids also appear to increase the rectal sensory threshold and the anal sphincter tone, resulting in harder stools and less frequent defecation[50]. Chronic opioid exposure can further disrupt intestinal permeability and induce gut microbiota dysbiosis through both-opioid receptors and toll-like receptor signaling, resulting in increased bacterial translocation, toxin accumulation, and pro-inflammatory signaling, which are associated with adverse outcomes in critically ill adults[49]. Centrally, opioids activate four receptor subtypes: Mu, delta, kappa, and opioid receptor-like-1. While central opioid receptors are primarily responsible for analgesia, central-opioid receptors, in particular, may also indirectly reduce gastrointestinal motility by altering autonomic outflow from the central nervous system[45,51].

Sedative agents such as benzodiazepines and propofol inhibits acetylcholine-mediated contractions of gastric and colonic smooth muscles, thereby predisposing to ileus[52,53]. Catecholamines such as epinephrine, commonly administered to critically ill adults, can slow the rate of gastrointestinal tract through beta-adrenergic stimulation, while dopamine has been shown to prolong small bowel transit[54,55]. The concomitant use of these agents can further exacerbate constipation.

Nutrition-related factors

Dehydration resulting from inadequate fluid administration or excessive diuretic use and the resultant hypovolemia may promote constipation[28]. Conversely, excess fluid resuscitation can result in splanchnic oedema, impairing gut motility [56]. Critically ill adults often receive nutrition enterally *via* a nasogastric tube or parenterally *via* intravenous routes. However, nothing by mouth status has been associated with constipation among critically ill adults[19]. Delayed initiation of enteral nutrition (EN) is a recognized risk factor. Although the American Society for Parenteral and EN (ASPEN) guidelines recommend initiating EN within 24-48 hours of ICU admission, timely initiation is frequently hindered in critically ill adults due to peristaltic disabilities caused by factors such as hypoxia and hypotension[11,57]. Delayed enteral feeding may reduce mechanical stimulation of peristalsis, exacerbating intestinal hypomobility that is often already present in critically ill adults. Additionally, low-fiber enteral formulas can compromise stool bulk formation, weaken the evacuation reflex and predispose patients to constipation[58].

Table 2 Contributing factors to constipation in critically ill adults

Category	Contributing factors	
Patient-related factors	Advanced age, prior abdominal surgery, immobility	
Disease-related factors	Systemic	Acute kidney injury, sepsis, shock
	Metabolic	Diabetes mellitus, hyper/hypothyroidism, electrolyte imbalances
	Neurological	Spinal cord injury, stroke, Parkinson's disease, multiple sclerosis, autonomic neuropathy
	Myopathies	Amyloidosis, scleroderma
	Psychological	Depression, anxiety, eating disorders
	Mechanical	Colorectal cancer, strictures, rectocele
Medication-related factors	Polypharmacy	
	Opioids	
	Sedatives	
	Anticholinergics, including antidepressants and antipsychotics	
	Antihypertensives, including calcium-channel blockers	
	Diuretics	
Nutrition-related factors	NPO status, delayed enteral feeding, inadequate fiber intake, dehydration	

NPO: Nothing by mouth.

CLINICAL IMPACT AND PROGNOSTIC IMPLICATIONS

As previously outlined, constipation in critically ill adults has multiple potential causes. Similarly, it has multiple clinical consequences (Table 3). Constipation can cause abdominal distension and discomfort, leading to upward diaphragmatic displacement and elevated intra-abdominal pressure. These changes reduce lung compliance and increase intrathoracic pressure, possibly resulting in respiratory complications such as atelectasis and pulmonary oedema[59]. Mostafa *et al*[14] reported that 42.5% of constipated ICU patients failed to wean from mechanical ventilation, compared to none in the non-constipated group. Supporting this, Gacouin *et al*[11] demonstrates that constipation in long-term ventilated ICU Patients is independently associated with higher rates of failure to wean from mechanical ventilation, increased risk of acquired bacterial infections and increased mortality.

Constipation may predispose critically ill adults to infectious complications. Fecal stasis due to constipation promotes overgrowth of gram-negative bacteria and increase in endotoxin, accompanied by a reduction of endogenous anaerobic and gram-positive flora[17]. Impaired peristalsis may cause progressive translocation of bacteria and endotoxins from gastrointestinal tract into systemic circulation, facilitating a systemic inflammatory response[60]. These changes contribute to higher rates of nosocomial infection, sepsis, multi-organ dysfunction and mortality among critically ill adults[17,61]. Systematically, constipation is associated with higher Sepsis-related Organ Failure Assessment and Acute Physiology and Chronic Health Evaluation II scores, indicating greater illness severity among ICU patients[17].

Constipation in critically ill adults is increasingly recognized not merely as a gastrointestinal motility issue but as a potential marker and contributor to acute intestinal dysfunction[59]. It often coexists with other motility disturbances, such as delayed gastric emptying, gastroparesis and ileus, which impair the initiation and progression of EN. Suboptimal nutritional support can compromise immune function, delay wound healing, reduce muscle strength, and prolong ICU stays, collectively contributing to worse prognosis[59]. Constipation may indicate impaired gut integrity and motility, and the gastrointestinal tract can act as a reservoir for systemic bacteremia, endotoxemia, or both in critically ill adults [62]. Severe dysmotility can lead to rare but serious complications such as intestinal pseudo-obstruction or bowel perforation which may necessitate surgical intervention[63].

Finally, constipation may have psychological and neurocognitive implications, including embarrassment, distress, and an increased risk of delirium in mechanically ventilated patients[3,64,65]. Given the adverse consequences of constipation on multiple organ systems, constipation may worsen critically ill adults' well-being[66].

INVESTIGATIVE STRATEGIES

Clinical assessment

A thorough bowel history is essential upon ICU admission to assess for potential constipation. However, as critically ill adults are often under sedation or mechanical ventilation, a focused abdominal examination and digital rectal examination (DRE) become essential bedside tools in evaluating suspected constipation. Abdominal examination may

Table 3 Possible complications associated with constipation in the intensive care unit

Category	Possible complications
Respiratory	Reduced lung compliance
	Prolonged mechanical ventilation and ICU length of stay
	Risk of VAP
	Pulmonary aspiration
Infectious	Bacterial overgrowth and translocation
	Intestinal microbiota disruption (dysbiosis)
	Systemic inflammation triggered by endotoxins and bacterial translocation
	Nosocomial infection and sepsis
Gastrointestinal	Abdominal distension and discomfort
	Elevated intra-abdominal pressure
	Vomiting
	Delayed gastric emptying
	Poor tolerance of enteral nutrition
	Risk of intestinal pseudo-obstruction or perforation
Neurocognitive and psychological	Risk of delirium
	Psychological distress due to constipation-related pain or discomfort
	Reduced patient comfort and well-being

ICU: Intensive care unit; VAP: Ventilator-associated pneumonia.

reveal distension or tenderness, while DRE allows for assessment of anorectal sensation, tone, coordination, and the presence of fecal impaction, stricture, obstruction, or blood. Importantly, DRE helps exclude mechanical causes that may require surgical treatment[67]. Additionally, the Bristol Stool Form Scale may be employed to classify stool consistency and guide diagnosis[13].

Laboratory investigations

A comprehensive laboratory assessment is essential for evaluating suspected constipation in critically ill adults, as metabolic and systemic disturbances frequently impair gastrointestinal mobility. Investigations include a complete blood count, renal function tests, and a basic metabolic panel[1]. Serum levels of potassium, magnesium, calcium and phosphate should also be measured, with prompt correction of any electrolyte imbalances, such as hypokalemia or hypomagnesemia[68]. In addition, thyroid function tests may be considered to exclude hypothyroidism which prolongs colonic transit time[69]. Inflammatory markers such as C-reactive protein may help identify critically ill adults at risk of constipation; elevated C-reactive protein levels by day 7 of ICU stay have been independently associated with delayed defecation[70].

Imaging

Imaging with X-ray or computed tomography is generally reserved for patients who failed to respond to initial treatment interventions or who present with signs suggestive of complications. An abdominal X-ray or computed tomography can confirm the presence of excessive bowel stool, colonic dilation or suspected ileus. Acute colonic pseudo-obstruction is a diagnosis of exclusion, where the abdominal X-ray will reveal a massively dilated colon, in particular the caecum and right colon[67]. Bedside point-of-care ultrasonography is increasingly performed to assess gastrointestinal function in ICUs[71]. Bedside point-of-care ultrasonography may reveal reduced or absent peristalsis, dilated bowel loops (> 5 cm), increased mucosal thickness and impaired intestinal wall perfusion - all indicative of fecal stasis associated with constipation[72,73].

TREATMENT STRATEGIES

Given the potential complications associated with constipation, treatment should prioritize the re-establishment and maintenance of regular bowel movements. This can be achieved through a combination of nutritional, pharmacological, and targeted approaches.

General nutritional approaches

Personalized nutritional therapy is essential in critically ill adults to optimize recovery and reduce complications, including constipation[74]. Key components of individualized EN strategy include nutrient composition - such as energy density, protein content, and fiber supplementation - as well as feeding strategies, including mode of delivery (continuous *vs* intermittent) and the site of tube feeding (gastric *vs* post-pyloric).

Concentration: Standard polymeric formulas are the most used EN formulations in critically ill adults, delivering macronutrients in non-hydrolyzed forms that simulate a typical diet. These formulas generally range from 1 kcal/mL to 2 kcal/mL in energy density and are formulated to meet 100% of the recommended dietary allowances for essential micronutrients within 1.0-1.5 L of intake. ASPEN guidelines recommend feeding between 12 kcal/kg and 25 kcal/kg in the first 7-10 days of ICU stay. Concentration of formulas to be delivered should also be tailored to the patient's energy requirements as metabolic demand may vary during different phases of critical illness[74]. Delivery of an optimal concentration of EN supports both nutritional adequacy and fluid management in the ICU setting, key factors in recovering gastrointestinal function.

In patients with fluid restriction, such as those with cardiac or renal failure, ascites, or syndrome of inappropriate antidiuretic hormone secretion, concentrated formulas (2 kcal/mL) can help achieve nutritional targets with limited fluid volumes[75]. However, gastric intolerance - such as increased gastric residual volumes or abdominal distension - should be closely monitored when using energy-dense formulas. In patients without fluid restriction, additional water flushes are often required to prevent dehydration and support bowel regularity, particularly when using hypercaloric feeds[75]. Nonetheless, current evidence on whether higher energy delivery improves gastrointestinal outcomes in critically ill adults remains inconclusive[76].

Protein intake: A protein dose of 1.2-2.0 g/kg/day is generally advised by international guidelines, with higher targets for critically ill adults with burns, obesity, or trauma[76,77]. A recent systematic review by Blaauw *et al*[78] reported that higher protein intake may be associated with fewer episodes of constipation in critically ill adults when compared to lower protein intake, although the certainty of this evidence remains low. Critically ill adults undergo pronounced catabolism, leading to rapid skeletal muscle loss that impairs functional capacity and metabolic health, including gastrointestinal function[74]. A recent randomized controlled trial (RCT) reported that high-protein regimens (1.8/kg/day) may help preserve muscle mass during the acute phase of critically illness, particularly when combined with early mobilization strategies[79]. However, a study by Chapple *et al*[80] revealed profound skeletal muscle anabolic resistance in critically ill adults, reducing their ability to utilize ingested protein for muscle protein synthesis despite preserved protein digestion and amino acid absorption. Moreover, a recent randomized clinical trial reported that augmented enteral protein intake did not significantly improve mortality or major clinical outcomes in critically ill adults[81]. These findings underscore the need to re-evaluate routine high-protein strategies in this population. Further high-quality studies are warranted to clarify the role of enteral protein provision on gastrointestinal outcomes, including constipation, in critically ill adults.

Fiber supplementation: Fiber supplementation in EN has been shown to accelerate whole gut transit time, benefiting enterally fed patients experiencing constipation due to slow colonic transit. In critically ill adults, dietary fiber also plays a broader physiological role - preserving gut barrier function, modulating immune responses, supporting the intestinal microbiota, and attenuating systemic inflammation[82]. Both soluble and insoluble fibers have been associated with enhanced gastrointestinal motility, reduced feeding intolerance, and shortened hospital length of stay[82]. Fiber is also shown to improve stool consistency at both extremes - softening hard, dry stools and firming loose, unformed stools, thereby promoting normalized bowel movements[58].

When selecting the type and quantity of fiber for enteral feeds, two main strategies are recommended: (1) Aligning with fiber intake levels observed in healthy populations; or (2) Tailoring the fiber formulation to achieve specific physiological outcomes relevant to the patient's condition[58]. However, caution is advised against using single-source fibers with extreme solubility or fermentability, as high doses of highly soluble fermentable fibers like inulin can cause flatulence, whereas exclusive use of insoluble fibers such as cellulose may exacerbate constipation[83,84]. The 2016 ASPEN and Society of Critical Care Medicine (SCCM) guideline supports the use of fermentable soluble fiber, such as fructo-oligosaccharides and inulin, for hemodynamically stable ICU patients receiving fiber-free enteral formulas[57]. However, the guideline advises against the routine use of mixed soluble and insoluble fiber preparations due to potential risks of bowel dysmotility and ischemia[57]. Insoluble fibers, which are poorly fermentable and contribute to stool bulk, may increase gastrointestinal workload. In critically ill adults with impaired gut motility or splanchnic hypoperfusion, insoluble fibers can contribute to delayed transit and ischemic complications[85]. Greater caution is thereby warranted in hemodynamically unstable or hypoxic patients, where it is important to reduce the proportion of insoluble fiber to avoid further bowel ischemia and injury[85].

Continuous *vs* intermittent mode of EN: The optimal mode of EN in critically ill adults remains debated, particularly between continuous (24-hour) and intermittent feeding. While the ESPEN guidelines recommend continuous feeding, a practice widely adopted in ICUs, this guidance is supported by limited high-quality evidence[77,86]. In comparison to continuous feeding, intermittent feeding more closely mimics physiological meal patterns and may better preserve circadian rhythms, which are often disrupted in critical illness[87]. A 2022 systemic review of 14 RCTs reported that continuous feeding was associated with a significantly higher risk of constipation compared to intermittent feeding, with no observed differences in mortality, aspiration, or feeding tolerance. While intermittent feeding reduced constipation risk, it was associated with higher incidence of diarrhea and abdominal distension[88,89]. Intermittent feeding may facilitate early mobilization by enabling scheduled fasting intervals and reducing the need for continuous connection to

feeding equipment. Further studies are needed to establish its clinical superiority.

Gastric tube *vs* post-pyloric feeding: The ASPEN-SCCM guidelines recommend gastric feeding as the route for EN due to its ease of placement, physiological alignment, and low invasiveness. Several RCTs have reported no significant differences in gastrointestinal complications or ICU mortality between gastric and post-pyloric feeding[90-92]. Conversely, recent meta-analyses suggest potential advantages of post-pyloric feeding over gastric feeding, which include a lower incidence rate of gastrointestinal complications, such as constipation, and reductions in the duration of mechanical ventilation, ICU stay, and hospitalization[93]. Despite these advantages, routine post-pyloric feeding is not currently recommended in the ICUs[77], as post-pyloric placement requires technical expertise and often relies on radiologic or endoscopic assistance which may delay feeding initiation. Additionally, post-pyloric feeding is considered less physiologic compared to gastric feeding and may be unsuitable in patients with lower gastrointestinal dysmotility. Post-pyloric access should be reserved for selected patients, particularly those with high aspiration risk or with gastric feeding intolerance due to gastroparesis[77,94].

General pharmacological approaches

Laxatives are often considered the first-line pharmacological treatment for constipation among critically ill adults. Enterally-administered laxatives can be broadly classified into osmotic laxatives, stimulant laxatives, stool softeners, and bulk-forming agents (Table 4). Patanwala *et al*[16] reported that critically ill adults given osmotic laxatives and/or stimulant laxatives were significantly more likely to have a bowel movement within the first 96 hours of ICU admission compared to those who did not. Osmotic laxatives can be further subdivided into saline and non-saline agents. Saline osmotic laxatives, such as milk of magnesia, magnesium citrate and sodium phosphate, act as hyperosmolar agents that increase stool water content and stimulate cholecystokinin, enhancing peristalsis[95]. However, their use is limited due to risk of hypermagnesia-induced paralytic ileus and hyperphosphatemia in patients with renal insufficiency or electrolyte disturbances[96]. Non-saline osmotic laxatives, such as lactulose and macrogol, are poorly absorbed polymers that draw water into the intestinal lumen, softening the stool[97]. Lactulose is among the most widely used agents for managing constipation and facilitates defecation without directly stimulating colonic motility; non-saline osmotic laxatives are thereby preferred for critically ill patients with suppressed peristalsis or impaired neuromuscular function. Lactulose is typically initiated at a dose of 10 mL twice daily and titrated up to a maximum of 20 mL three times daily[14].

A randomized controlled trial involving 308 ICU patients who had not defecated by day 3 of admission studied the relative efficacy of osmotic laxatives. Patients were randomized to receive either lactulose, macrogol (polyethylene glycol), or placebo three times daily for up to four days. Both lactulose and macrogol were equally effective in promoting bowel movements, with around 75% of patients in each treatment group having successful defecations, compared to only 31% in the placebo group. Macrogol was associated with a lower incidence of acute intestinal pseudo-obstruction, while lactulose was linked to a shorter ICU length of stay[98]. In managing OIC, macrogol appeared more effective than lactulose and was associated with fewer adverse effects.

Stimulant laxatives such as senna and bisacodyl are prodrugs that are hydrolyzed into active metabolites by colonic bacteria (senna) and intestinal and colonic brush border enzymes (bisacodyl), which exert anti-absorptive, secretory, and prokinetic effects[99]. Patanwala *et al*[16] reported that senna was strongly associated with occurrence of a bowel movement in critically ill adults, and bisacodyl showed a trend towards statistical significance. The safety profile of senna is generally favorable for short-term use in the ICU, but adverse effects such as abdominal cramping and diarrhea may occur at higher doses[16].

Bulk-forming laxatives, such as psyllium and methylcellulose, are commonly used as first-line agents in ambulatory patients. However, they may be less suitable for critically ill adults who require rapid relief from constipation due to their delayed onset[100]. Moreover, both bulk-forming laxatives and stool softeners rely on adequate fluid intake to be effective, which is often difficult to achieve in the ICU setting[16]. Bulk-forming laxatives should be avoided in patients with fluid restriction, limited mobility, gastrointestinal strictures or partial obstruction, as they may increase the risk of fecal impaction[101].

Suppositories and enemas are typically reserved for relieving constipation when oral laxatives are ineffective[4]. Laxative suppositories exert their effects primarily through local stimulation of the rectum and may be ineffective when feces are predominantly in the colon or more proximal segments of the gastrointestinal tract[100]. Excessive use of enemas may result in rare but serious bowel perforation or hyperphosphatemia[102].

Prokinetic agents such as erythromycin and metoclopramide have been shown to enhance gastric emptying and improve feeding intolerance in critically ill adults[103]. Critically ill adults are particularly susceptible to gastroparesis and intestinal pseudo-obstruction, for which erythromycin is often considered the prokinetic agent of choice due to its motilin receptor-mediated effects on upper gastrointestinal motility[104]. While these agents are occasionally used alongside laxatives, their prokinetic activity is largely confined to the stomach and small intestine, with minimal impact on colonic motility[16]. Prokinetic therapy has not been shown to significantly reduce rates of pneumonia, mortality or ICU length of stay, nor does it appear to increase the risk of diarrhea[105]. Furthermore, most studies predominantly evaluate the role of prokinetic agents in reducing gastric residual volumes rather than directly addressing constipation [106]. Prokinetic agents are generally not advised as first-line treatment for constipation due to associated risks such as cardiac side effects and the potential for antimicrobial resistance[107].

Specific approaches for various causes

Initiation of a laxative regimen is generally recommended for patients started on opioid therapy to prevent the development of OIC[97]. While laxatives are generally well tolerated, their prolonged use may lead to adverse side effects

Table 4 Enterally-administered laxatives used in management of constipation in the intensive care unit

Parameter	Osmotic laxatives		Stimulant laxatives	Stool softeners	Bulk-forming laxatives
	Saline	Non-saline			
Examples	Milk of magnesia; magnesium citrate; sodium phosphate	Lactulose; macrogol/PEG	Senna; bisacodyl	Docusate	Psyllium; methylcellulose; polycarbophil
Mecha-nism of action	Increase intestinal osmotic pressure and water retention; promotes intestinal motility by stimulating cholecysto-kinin secretion	Poorly absorbed by the gut and act as hyperosmolar agents	Increase intestinal motility, water and electrolyte secretions; stimulates peristalsis	Act as a surfactant which emulsifies stool with fat and water	Increase stool bulk and frequency
Onset	0.5-6 hours (rapid)	Lactulose: 24-72 hours; PEG: 24-96 hours	6-12 hours	24-72 hours (slow)	12-72 hours (slow)
Clinical use	Rescue treatment for acute constipation. May cause electrolyte disturbances; caution in renal or cardiac dysfunction	Common use in ICU. Lactulose may result in shorter ICU length of stay; PEG may be more effective in opioid-induced constipation and has lower risk of acute intestinal pseudo-obstruction	Common use in ICU. Often combined with osmotic laxatives	Prophylactic use only. Often adjunctive, limited efficacy when used alone	Rarely used in ICU. Requires adequate hydration. Contraindicated in presence of bowel obstruction, ileus or megacolon

PEG: Poly-ethylene glycol; ICU: Intensive care unit.

such as nausea, vomiting, diarrhea, and abdominal pain, potentially resulting in treatment discontinuation[5]. Moreover, as laxatives do not address the underlying pathophysiological mechanisms of OIC, symptom relief may only be achieved in a proportion of patients[108].

Peripherally acting mu-opioid receptor antagonists (PAMORAs), such as methylnaltrexone, naldemedine, and naloxegol, have emerged as a promising therapeutic class for OIC. Sawh *et al*[109] reported that methylnaltrexone, a selective PAMORA, demonstrates greater efficacy than conventional laxatives in treating OIC. Importantly, it is well tolerated and crosses the blood-brain barrier only minimally, thereby preserving central opioid analgesia. Common adverse effects of PAMORAs include abdominal pain, nausea and diarrhea[97]. Concerns have been raised regarding the risk of intestinal perforation with post-operative use of methylnaltrexone[110]. The American Gastroenterological Association recommends traditional laxatives as first-line therapy and endorses PAMORAs in patients requiring escalation of therapy. In laxative-refractory OIC, the American Gastroenterological Association specifically recommends naldemedine or naloxegol, followed by methylnaltrexone, over no treatment. However, these recommendations are based on general patient populations, and their applicability to critically ill adults remains uncertain[97].

Naloxone is a primarily a centrally acting receptor antagonist that readily crosses the blood-based barrier; however, when administered orally, it undergoes extensive first-pass hepatic metabolism, resulting in low systemic bioavailability and confines its activity largely to intestinal opioid receptor[108]. Standard oral doses of naloxone are initiated at 4 mg/day and titrated up to 18 mg/day and may alleviate refractory OIC[100]. Higher oral doses, however, may pass the blood-brain barrier, carrying a risk of systemic reversal of opioid analgesia. Such doses may also precipitate withdrawal symptoms such as yawning, sweating and shivering, particularly in patients with underlying hepatic dysfunction, where more naloxone enters systemic circulation and antagonizes central opioid receptors[111-113].

The use of oral naloxone in fixed-dose combinations with oxycodone, a centrally acting opioid receptor agonist, has also generated clinical interest, although existing studies primarily involve patients with chronic pain and do not evaluate its use in critically ill populations. Prolonged-release oxycodone/naloxone combinations (2:1 fixed ratio) were evaluated in four RCTs in 974 patients with chronic cancer or non-cancer pain[114-117]. The trials reported that switching from other opioids to an oxycodone-naloxone combination was generally safe, effective and associated with fewer OIC-related complications[116]. In a retrospective analysis, Habeeb *et al*[118] evaluated critically ill adults who received either enteral naloxone or subcutaneous methylnaltrexone while on continuous opioid infusions for at least 48 hours. Naloxone was seen to significantly shorten the time to first defecation compared to methylnaltrexone (a median of 18 hours *vs* 41 hours). On the other hand, methylnaltrexone was generally better tolerated, as it acts peripherally without reversing desired central opioid analgesia or precipitating opioid withdrawal, regardless of its route of administration[119]. Future high-quality, prospective trials are warranted to evaluate the relative efficacy and applicability of μ-opioid antagonists in the ICU setting.

Critical illness-related colonic ileus, characterized by the absence of stool passage despite normal gastric emptying and unremarkable physical and radiological findings, may be effectively managed with a continuous intravenous infusion of neostigmine at a dose of 0.4-0.8 mg/hour[67,120]. Neostigmine, a peripherally acting acetylcholinesterase inhibitor, has also demonstrated efficacy in severe, refractory cases of acute colonic pseudo-obstruction, particularly when conservative measures have failed (increased distension, increasing fecal diameter or fecal diameter > 10 cm)[121,122]. Neostigmine has also demonstrated effectiveness for patients who do not respond to initial laxative treatment[20]. However, its administration necessitates continuous cardiac monitoring due to its adverse effects, including bradycardia and arrythmia, or in rare cases, bowel perforation[123,124]. Erythromycin has also shown to be effective in treating colonic

pseudo-obstruction in a few case reports[125,126].

In the management of constipation associated with neurogenic bowel dysfunction, understanding the patient's baseline bowel frequency prior to neurological injury is essential for guiding an individualized bowel program[127]. For patients with upper motor neuron lesions, reducing dietary fiber - particularly insoluble fiber - can help achieve a stool consistency that facilitates evacuation following rectal stimulation. Osmotic laxatives and stool softeners may be used concurrently to support stool softening. In contrast, patients with lower motor neuron lesions may benefit from increased fiber intake to improve stool bulk and consistency, with bulking agents and osmotic laxatives or stool softeners often used in combination[127]. Additionally, physical interventions such as abdominal massage, peri-anal and anorectal digitation have been shown to be effective in facilitating bowel evacuation[128,129]. A treatment algorithm for neurogenic bowel dysfunction has also been outlined by the International Consultation on Incontinence to aid clinicians in delivering lesion-specific care[130]. Emerging interventions targeting brain-gut axis, including prokinetic agents and microbiota-modulating therapies, have shown efficacy in chronic constipation. Prokinetic agents such as prucalopride, a selective 5-hydroxytryptamine receptor 4 agonist, act through 5-hydroxytryptamine receptor 4 receptors in the gastrointestinal tract and has demonstrated efficacy in improving colonic motility in chronic idiopathic constipation among the general population[98,131]. Fecal microbiota transplantation had an improvement rate of 64.8% in chronic constipation[132]. However, evidence in critically ill adults is lacking, and the safety and efficacy of these therapies in the ICU setting remain to be established.

PREVENTIVE STRATEGIES

Prophylactic laxative administration

Prophylactic administration of laxatives appears beneficial in critically ill adults[13]. In a case-control study led by Guardiola *et al*[12], prophylactic administration of macrogol to 197 critically ill adults on the first day of admission led to better outcomes compared to initiating treatment on day 4. Stool softeners such as docusate are ideal for preventing constipation but are generally ineffective when used alone for established cases[16,21].

Mobilization and manual techniques

Where applicable, early mobilization strategies, including passive exercises of the lower limbs and trunk, should be implemented for bed-bound patients. passive exercises of the lower limbs and trunk contributes to mechanical stretching of the intestinal tract and stimulation of parasympathetic nervous system, facilitating intestinal smooth muscle contraction[133]. A 15-minute abdominal massage, performed twice daily for three consecutive days in an RCT was also reported to facilitate early defecation and lower the prevalence of constipation in critically ill adults[134].

Minimizing constipating medication exposure

Preventing constipation in critically ill adults includes minimizing exposure to constipating medications, including opioids, anticholinergics and calcium channel blockers. Routine re-evaluation of the indication, dosage, and duration of these agents should be performed. The SCCM recommends multimodal analgesic strategies that incorporate non-opioid analgesics to minimize opioid use[135,136]. Nefopam, for instance, may be considered as an adjunct or alternative to opioids, as it does not adversely affect intestinal motility, vigilance, or respiratory drive. In contrast, routine use of IV lidocaine infusions or cyclooxygenase-1 selective non-steroidal anti-inflammatory drugs are discouraged in critically ill adults due to limited data on safety profile and potential systemic adverse effects[136,137]. Notably, lidocaine has not been shown to reduce time to first defecation. These recommendations are based on low or very low-quality evidence, and larger sized studies in critically ill populations are required to clearly evaluate the opioid-sparing properties of non-opioid analgesics and their ability to reduce opioid-related adverse effects, including constipation[136].

Resolution of critical illness

Resolution of underlying critical illness is fundamental to the recovery of gastrointestinal function. As physiological stability improves - such as with recovery from shock, hypoxic respiratory failure, or sepsis - intestinal perfusion, motility and enteric nervous system function may also recover, thereby reducing the risk of constipation[138].

Perioperative measures

Post-operative constipation can be mitigated through several evidence-based perioperative strategies. Minimizing perioperative opioid use through multimodal pain management, avoiding routine use of nasogastric tubes and abdominal drains, early removal of urinary catheters to facilitate mobilization, and meticulous perioperative fluid and electrolyte management are key strategies that have been shown to reduce the incidence of post-operative ileus[139]. In colorectal surgery, avoidance of prolonged fasting and early mobilization are essential components of Enhanced Recovery After Surgery protocols[139].

BOWEL MANAGEMENT PROTOCOLS

Given the frequent occurrence of constipation among critically ill adults, the adoption of a bowel management protocol (BMP) to achieve a standardized, systematic approach to bowel care has been highlighted in several studies[4,65].

Successfully BMP implementation has been associated with measurable improvements, including increased documentation rates and enhanced quality of care[4,140]. Ritchie *et al*[141] demonstrated that when effectively adopted, BMPs reduced constipation prevalence from 83% to 40%.

However, the implementation of standardized BMPs remains inconsistent across ICUs. Evidence from a nursing survey conducted across 44 ICUs in New South Wales, Australia, revealed that only 32% of respondents were aware of a routine BMP in their ICUs, and of these, just half reported universal implementation across all patients[5]. Similar findings have been reported in the United Kingdom, where fewer than 20% of surveyed ICUs had a formal 'bowel care guideline' in place[142]. A study by Knowles *et al*[143] reported that following the formal introduction of BMP, 66% of clinicians did not adhere to the protocol.

Barriers identified include ambiguity of protocol content, reluctance to change practice, limited knowledge of procedures such as DRE, and low prioritization of constipation[104,144,145]. Healthcare providers' knowledge gaps, attitudes, and beliefs have been shown to deter compliance with established BMPs, highlighting the need for targeted implementation strategies that address behavioral and educational barriers[143].

Furthermore, a content analysis by Dionne *et al*[146] reported that 33 of 44 ICUs had at least one bowel protocol, yielding a total of 37 BMPs. Among these BMPs, 59.4% were standalone documents, while the remainder were incorporated into ICU admission orders. Substantial variability was observed among the BMPs, with initiation, escalation, and discontinuation criteria often inconsistently defined or applied across institutions. Notably, none of these BMPs addressed prophylactic laxative use, fiber supplementation, or strategies to minimize opioid use, reflecting a general lack of focus on preventive measures[146].

Such inconsistency in implementation and criteria across BMPs has significant clinical implications, as it may delay recognition of constipation and hinder timely intervention in the ICU[140]. Such discrepancies may compromise optimal management of constipation in critically ill adults. A systematic review and meta-analysis by Oczkowski *et al*[147] highlighted the scarcity of rigorous research to support the clinical efficacy of BMPs, with limitations largely due to the small number of studies and their methodological heterogeneity. This underscores the urgent need for rigorous, large-scale randomized controlled trials to determine the optimal class, dose and timing of interventions. Such evidence is essential to guide the revision and standardization of existing BMPs, many of which remain outdated, inconsistent, and lacking empirical support. In the interim, Table 5 presents a suggested approach to the management of constipation in critically ill adults, incorporating both preventive and therapeutic elements[20,146]. This tiered approach draws upon current literature and commonly adopted clinical practices to delineate actions based on timing since last bowel movement, while also accounting for diagnostic evaluation and escalation criteria.

FUTURE DIRECTIONS

Identifying critically ill adults at highest risk of constipation remains an unmet need. Research into biomarkers or risk stratification tools to anticipate constipation in the ICU could enable earlier and more targeted interventions.

In terms of management, high-quality evidence guiding pharmacological and non-pharmacological management remains sparse, with available studies often limited by small sample sizes, single-center designs, and observational methods. Insufficient data exist to determine the optimal timing, dosing, and combinations of laxatives, as well as their effect on patient-centered outcomes including duration of mechanical ventilation, ICU length of stay, and gastrointestinal complications. Future trials should directly compare prophylactic *vs* reactive laxative regimens to clarify the most effective timing of intervention. Further comparative evaluation of non-osmotic agents, such as stool softeners, stimulant laxatives and opioid antagonists, is also warranted**.** PAMORAs have shown promise in promoting defecation in OIC, but their efficacy and safety should be validated through well-designed, large-scale prospective ICU trials[148].

Emerging interventions targeting the brain-gut axis, such as microbiota-modulating therapies, may offer potential to prevent or mitigate constipation, particularly in critically ill adults with neurological impairment. These therapies, including probiotics, prebiotics, and fecal transplantation, should be systematically evaluated, particularly in patients with disrupted gut microbiota or neurological dysfunction. Finally, structured BMPs are widely used but vary considerably in criteria and implementation across ICUs; rigorous multicenter RCTs are needed to clarify their impact on constipation and broader ICU outcomes. Addressing these research gaps is essential to develop ICU-specific evidence-based BMPs that improve both clinical outcomes and quality of care.

CONCLUSION

In critically ill adults, constipation extends beyond being a mere prognostic marker and may reflect an underlying organ dysfunction that warrants timely recognition and management. Although a direct causal link between constipation and subsequent organ failure remains uncertain, appropriate treatment may contribute to earlier restoration of bowel dysfunction and reduce complications associated with constipation. Despite its high prevalence and clinical significance, constipation remains under-recognized and inconsistently addressed in the ICU. Given its multifactorial etiology, a comprehensive and individualized approach is warranted. Key preventive strategies include early initiation of EN, early mobilization, and judicious use of constipating medications, with preference for multimodal analgesia to minimize opioid exposure. A more consistent implementation of bowel management protocols, alongside evidence-based nutritional and treatment strategies, can support earlier identification and targeted intervention among critically ill adults.

Table 5 Suggested approach to management of constipation in the intensive care unit

Phase/criteria	Management	
On admission (< 24 hours)	Patient history: Frequency of bowel movements, stool consistency, timing of last stool, baseline laxative use, history of bowel disorders	
	Identify risk factors: Opiate use, immobility, neurological impairment, surgery, *etc.* Minimize risk factors where possible	
	Preventive strategies	Correct fluid and electrolyte imbalances (potassium, calcium, magnesium)
		Ensure adequate hydration
		Initiate early enteral nutrition
	Consider initiating daily prophylactic laxatives in high-risk patients	
> 24 hours since last defecation	Review and minimize constipating agents (*e.g.* opioids, anticholinergics)	
	Perform abdominal and rectal examination	
	Start first-line laxative therapy (*e.g.* lactulose, PEG, senna, bisacodyl); increase dose if already on therapy	
	Reassess and adjust therapy daily	
> 48 hours since last defecation (escalation phase)	Repeat rectal exam to assess impaction	
	Consider	Abdominal X-ray to evaluate for ileus or obstruction
		Combination/adjunctive therapy
		Rectal interventions (*e.g.* enemas)
Refractory constipation	Consider advanced investigations and interventions	Abdominal CT to rule out mechanical obstruction
		Neostigmine (especially in suspected acute colonic pseudo-obstruction)
		Manual rectal dis-impaction if indicated
		Surgical decompression as last resort
Discontinuation criteria	Presence of diarrhea or resolution of symptoms	
Contraindications to bowel management protocol	Renal disease	
	Major abdominal surgery/bowel obstruction	
	Neutropenia (or bone marrow transplant)	
	Nausea and vomiting, undiagnosed abdominal pain	
	Thrombocytopenia	

CT: Computed tomography; PEG: Poly-ethylene glycol; ICU: Intensive care unit.

FOOTNOTES

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